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The effects of naltrexone on repressive coping and disclosure of emotional material : a test of the opioid-peptide theory of repression/hypertension

Jarred Wayne Younger

To the Graduate Council:

I am submitting herewith a dissertation written by Jarred Wayne Younger entitled "The effects of naltrexone on repressive coping and disclosure of emotional material : a test of the opioid-peptide theory of repression/hypertension." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Psychology.

Kathleen Lawler, Major Professor

We have read this dissertation and recommend its acceptance:

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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THE EFFECTS OF NALTREXONE ON REPRESSIVE COPING AND DISCLOSURE OF EMOTIONAL MATERIAL: A TEST OF THE OPIOID-PEPTIDE THEORY OF REPRESSION/HYPERTENSION.

A Dissertation
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Jarred Wayne Younger
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Abstract

A relationship between hypertension and repression has long been observed in the psychological literature. Those who exhibit repressive coping styles also tend to exhibit elevated blood pressure levels. Classically, this relationship has been explained by emphasizing the role of repression in elevating, through unknown mechanisms, blood pressure. Recent research, however, suggests that the directionality of this relationship may be reversed, and high blood pressure, through baroreceptor-mediated, endogenous-opioid activity, may result in repressive phenomena. The present study tests this hypothesis by comparing the disclosure of 8 normotensives and 10 hypertensives after receiving either the opioid antagonist naltrexone or a placebo. Administration of the drug or placebo was double-blind and randomized across 4 laboratory visits. Following absorption of the drug, participants verbally described their mood, conflicts, stressors, and concerns. Following the verbal narrative, participants completed measures of repressive coping style, disclosure, mood, and self-perceptions. To accommodate for the small sample size, planned comparisons were used for analyses. Specifically, the hypothesis was tested that naltrexone would significantly alter dependent measures, in the direction of higher disclosure and less repression, in hypertensive subjects only. Results supported the hypothesis. Significantly greater disclosure and significantly less repression were exhibited by hypertensives in the drug sessions versus placebo sessions. The drug condition had no effect on the self-report of normotensives. The results provide initial evidence for the role of endogenous opioids in repression and hypertension.

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CHAPTER I: INTRODUCTION

A link between the psychological concept of repression and the physiological condition of hypertension is well described in the psychosomatic literature. These two phenomena have been found to be positively correlated, with those showing signs of repression also exhibiting elevated blood pressure or hypertension. This relationship has been a key example in the academic problem of the mind/body relationship and the apparent correlation between repression and hypertension has been a central argument for the notion of psychosomatic illness and the idea that complex psychical processes can have concrete, physical consequences.

The relationship between repression and hypertension has been strongly supported by the scientific literature. A number of correlational studies have found an inverse relationship between self-reported stressors and hypertension. In one study, Winkleby, Ragland, and Syme (1988) conducted a comprehensive health exam on 1,428 San Francisco bus drivers. In addition to self-report stress scales, measures of blood pressure (as part of a physical exam) were included. The authors found a significant, inverse relationship between the bus drivers' blood pressures, as measured in the checkup, and their self-reported, daily stressors. Bus drivers with the highest blood pressure levels were least likely to report current stress. This inverse relationship has been found in many other studies as well. Rose, Jenkins, and Hurst (1978) found that hypertensive air traffic controllers reported significantly fewer life-change events, as compared to their normotensive coworkers. Harburg and colleagues (1970) found that hypertensive members of inner-city Detroit communities reported their environment as less dangerous than did normotensives in the same communities. Similarly, Tibblin &

Lindstrom (1972) found that hypertensives reported fewer health symptoms and that degree of hypertension was negatively correlated with symptom reporting.

The results of these studies are most striking in how they differ from the rather large body of evidence suggesting a positive relationship between stressors and blood pressure. Environmental stressors have been linked to elevated blood pressure in a variety of situations and populations, including catastrophic disasters (Baum, 1990), dangerous living areas (Rofe & Goldberg, 1983), job loss (Kasl & Cobb, 1970), and job strain (Theorell et al., 1991; Fox, Dwyer, & Ganster, 1993; Schaubroeck & Ganster, 1993). A meta-analytic review of psychosocial stressors and hypertension/elevated blood pressure concluded that the difference could be accounted for by objective versus subjective measures of stress (Nyklicek, Vingerhoets, & Van Heck, 1996). When objective indices of stress were used (e.g., laboratory stressor, high-risk job, or residence in a dangerous neighborhood), a strong positive correlation between stress and blood pressure can be seen. This relationship, however, is non-existent or even inverted when self-report scales are used as a measure of stress. This discrepancy between self-report and objective measures of stress and their relationship to blood pressure is hypothesized to be the result of an altered basis of stress appraisal exhibited by hypertensives (Nyklicek, Vingerhoets, & Van Heck, 1996; Winkleby, Ragland, & Syme, 1988). Hypertensives neither acknowledge nor disclose stress to the degree that normotensives do.

Evidence of an altered basis of perception in hypertensives was offered by Sapira, Scheib, Moriarty & Shapiro (1971). In their study, normotensive and hypertensive individuals were shown two movies: one of a "good doctor" interacting with a patient and one of a "bad doctor," who was very rude with the patient in the film. Hypertensive

individuals, who exhibited greater mean arterial pressure responses to the films, were nevertheless much more likely than normotensives to report seeing no differences in the behavior of the two doctors.

Hypertensives also report fewer life events and less emotional reaction to those life events. Svensson and Theorell (1983) found that hypertensives exhibiting consistent vasoconstriction (but not other hypertensives) reported fewer life events and less emotional reaction, when compared to normotensive and hypotensive groups. Similar results have been found in longitudinal studies. Theorell, Svensson, and Waller (1986) measured the blood pressure of 106 men at age 18. Ten years later, those subjects who had earlier exhibited early stage (and asymptomatic) hypertension reported significantly fewer life events than the other subjects. The diminished reporting exhibited by hypertensives also extends to self-reported neurotic symptoms (Davies, 1970) as well as self-reported cold-pressor (McCubbin & Bruehl, 1994), finger-pressure (Bruehl, Carlson, and McCubbin, 1992) and thermal (Sheps, Bragdon, Gray, Ballenger, Usedom, & Maixner, 1992) pain.

Zamir and Shuber (1980) tested the relationship between blood pressure and the experience of pain in hypertensive and normotensive individuals. Electrical stimulation of tooth pulp was gradually increased until the subject reported being in pain. Hypertensive status significantly predicted reported pain threshold in three separate age groups. In each case, hypertensives withstood almost double the voltage of the normotensive group. Furthermore, there were significant positive relationships between blood pressure (systolic and diastolic) and pain threshold in all groups. Increased tolerance to pain has also been found in adolescent boys with subclinical elevations in

resting systolic blood pressure, suggesting that the inverse blood pressure/pain reporting relationship begins before the onset of hypertension (Ditto, Seguin, Boulerice, Pihl. & Tremblay, 1998). Laboratory studies have also found that the reduced self-report of pain can be extended to psychosocial stress as well (Nykilcek, Vingerhoets, & Van Heck, 2001).

While hypertensives have been found to exhibit *repression-like* behaviors, attempts to measure repression, *per se*, has proven difficult. Most early laboratory work on repression was criticized as being poorly defined or too far removed from the repression encountered by psychotherapists (Davis & Schwartz, 1987). Objective measures of a repressive coping style, however, served as a catalyst for much of the laboratory work on repression. Byrne (1961) produced the Byrne Repression-Sensitization (R-S) scale to differentiate the two coping styles. More recently, however, Weinberger and others have proposed a measure of repressive coping style that crosses two existing scales: anxiety and defensiveness (Weinberger, Schwartz, & Davidson, 1979). Those exhibiting the "repressive coping style" score low on state anxiety (as measured, for example, by the Taylor Manifest Anxiety Scale; Taylor, 1953) but high on defensiveness, as measured by the Marlowe-Crowne Scale of Social Desirability (Crowne & Marlowe, 1960). Repressive copers are, therefore, those who claim low anxiety but score high on defensiveness, indicating that they may have a tendency to underreport negative experiences.

Research utilizing this method of identifying repressors has been fruitful, with many studies showing that repressive groups exhibit physiological stress responses greater than either true high anxious or true low anxious groups (Aspendorf & Scherer,

1983; Weinberger, 1990). Furthermore, defensiveness (measured by the Marlowe-Crowne Scale) has been found to be strongly associated with hypertension, regardless of high or low anxiety scores (Mann & James, 1998).

A review of psychosocial variables and hypertension found support for a repression/hypertension link (Sommers-Flanagan & Greenberg, 1989). Of nine studies looking at repression-type variables, six found a significant relationship between repression and hypertension. The wide range of scales used to measure "repression" make results difficult to compare, however. The Thematic Apperception Test, MMPI Alexithymia Scale, Byrne's Repression-Sensitization Scale, Crowne-Marlowe Social Desirability Scale, and General Well-Being Questionnaire were all used to measure denial of negative affect or neuroticism.

Previous research, therefore, supports the notion that repression and hypertension are closely related. Increases in blood pressure are associated with diminished negative self-report. Individuals with hypertension or elevated blood pressure report less pain, stress, neurotic symptoms, and negative affect than do normotensives. The next step, then, after determining the existence of a relationship, is to ascertain the directionality of that relationship.

The classic view of psyche-soma issues was put forth by early clinicians and psychoanalysts who believed that repression acted as the causal variable. One of the earliest recorded suggestions of a casual relationship between psyche and soma was offered by a tenth century physician, who believed that repressed hostility was a direct cause of rheumatoid arthritis (Shafii, 1973; as cited in McMahon, 1976). It was Freud and Breuer, however, who clarified and popularized the first unidirectional model of

psychosomatic phenomena. In their (1893/1966) hydraulic model of emotional functioning, traumatic events result in excess psychic energy that must be released. If this energy is not released, it will accumulate and eventually find another form of expression, either through physical or psychological symptoms (Smyth and Greenberg, 2000). Many current theorists and clinicians still maintain that distress, when not allowed to manifest psychically, finds a somatic route of expression (McDougall, 1989).

The greatest problem with the early model of disease as physical expression of psychological distress lies perhaps in the inability of the theory to fit known physiological and neurological mechanisms. There are no known physiological systems that support the idea that dammed up emotional energy can find a route of expression through the body and thus cause physical disease. There are no convincing biological, physiological, or neurological models which explain 1) the physical form of this energy, 2) how the energy is blocked or 3) how psychical energy transmutes to a physical one.

Partially in response to these problems, modern psychodynamic theory has replaced the hydraulic model with a different hypothesis: the effort required to maintain psychological repression (i.e., to actively keep offending material out of conscious awareness) puts a constant strain on the physiological system. Overactivation of the physical body is necessary to provide the energy required to maintain this psychological dissociation (Esterling, Antoni, Kumar & Schneiderman, 1990). With this approach, there is no need to hypothesize possible points where bad psychical energy is transmuted into bad physical energy. Repression can be described as simply another cognitive/mental process that requires energy. The current psychodynamic view of psychosomatic phenomena is captured by Shedler, Mayman, and Manis (1993), who state,

"Psychological defense has physiological costs. It is associated with autonomic reactivity and may be a risk factor for medical illness."

Since blood pressure is closely tied with activation of the sympathetic nervous system, which provides and mobilizes energy for most behavior, its place in the "physiological cost" hypothesis can be described thusly: chronically elevated blood pressure levels are the result of resources being constantly utilized for the maintenance of repression. Because the anxiety-provoking thoughts can never be allowed to enter consciousness, the demand on the cardiovascular system is constant. Hypertension, then, is simply the manifestation of the sympathetic system being constantly overworked in order to fuel the active and ongoing repression.

Very little evidence, however, exists for this view of repression as the causal variable in the repression/hypertension relationship. Much of the support comes from case studies in which a drop in blood pressure was observed after the disclosure of some traumatic event (Mann and Delon, 1995). These studies are usually problematic, however, in that the frequency of cardiovascular measurement is not sufficient to clearly determine whether the drop in blood pressure or the reduction of repression came first.

Limited evidence for the repression-causes-hypertension model can be found in the small but suggestive emotional suppression literature. In these studies, emotional suppression (the *conscious* inhibition of one's own emotional expressive behavior) is experimentally varied by instructing a group to show or not to show any emotion during a lab stressor, regardless of their natural inclinations. In an early study by Gambaro and Rabin (1969), greater blood pressure increases were induced in subjects who were not allowed to express anger to an anger-arousing lab stressor. In a more recent experiment,

Gross and Levenson (1993) instructed subjects to watch disgust-evoking films: one of a burn victim treatment and another of an amputation procedure. After determining that these films elicited strong feelings of disgust in viewers, the films were shown to a nonsuppression group and a suppression group. The suppression group was instructed to behave "in such a way that a person watching you would not know you were feeling anything." In general, the suppression group demonstrated a significantly different physiological response to the film in a way that suggested increased sympathetic arousal (greater skin conductance, greater finger pulse amplitude, and shorter pulse transmission times to the finger).

The study's usefulness to the present discussion, however, is limited by several factors. First, blood pressure, which is a necessary variable for discussing hypertension, was not measured in the Gross and Levenson (1993) study. Second, the only cardiovascular measure related to blood pressure that was included in the study was heart rate, and the suppression group exhibited greater *deceleration* as compared to the non-suppressing group. This finding runs counter to what would be expected if suppression was causally linked to elevated blood pressure.

A more recent study (Harris, 2001) addressed one of the previous arguments by including blood pressure in the physiological variable set. Participants were instructed to sing "The Star Spangled Banner" in front of a video camera. Afterwards, each subject watched the videotape of their singing with two confederates (this process was demonstrated previously by the same author to produce strong feelings of embarrassment). Similar to the Gross & Levenson (1993) study, one group was instructed, before the confederates entered the room, not to display any emotion and to

behave so that no one could guess what emotion they were experiencing. Results showed that the suppression group demonstrated significantly higher systolic and diastolic blood pressure as compared to the nonsuppression group. In addition to its inclusion of blood pressure, this study is important because it measured emotional suppression in a live interpersonal (albeit contrived) situation. This study shows that emotional inhibition can have an impact on blood pressure.

Unfortunately, all these studies share a common limitation: that the experimental group was given a task (to hide emotions) above and beyond that of the control group. Increased blood pressure, then, may be due mainly to the anxiety of being evaluated or the effort required to perform the task (Gross & Levenson, 1993). It is unknown, therefore, whether or not suppression has any effect above and beyond that of any other task in which the participant might be evaluated.

Furthermore, and more important to the present discussion, the authors in the previous studies have made no attempt to link their findings to *unconscious* repressive phenomena. With the available evidence, it seems more parsimonious to assume that differences in suppression and nonsuppression groups exist as a natural function of a conscious task. The results, therefore, should not be used to suggest that unconscious repression of emotions results in similar cardiovascular reactivity.

As the last form of support, disclosure has been investigated as a factor in blood pressure levels. If hypertension is a result of psychological repression, inhibition, or suppression of traumatic events or anxiety-provoking conflict, then the release of traumatic memories through disclosure should lead to a decrease in blood pressure. While a number of studies have shown that disclosure leads to lower physician visits

(Pennebaker & Beall, 1986; Greenberg & Stone, 1992) and increased immune function (Pennebaker, Kiecolt-Glaser, & Glaser, 1988; Francis & Pennebaker, 1992; Esterling et al., 1990), no study has found any reduction in blood pressure.

Pennebaker, Hughes, and O'Heeron (1987) investigated physiological differences between high and low disclosure. Participants talked about two subjects: 1) a traumatic experience and 2) what they were going to do after the experiment. Individuals were categorized as either high or low disclosers based on judged depth of disclosure in their spoken narratives. Following the traumatic-event narrative, SBP levels of low-disclosers returned to baseline levels, while SBP levels of high-disclosers dropped to levels significantly lower than baseline levels. These results were interpreted by the authors as evidencing greater recovery facilitated by high disclosure.

Interpretation of the data is complicated, however, by a number of factors. First, the main independent variable, disclosure, was an unassigned attribute variable. Differences in physiology, therefore, may be due to trait correlates of a high tendency to disclose. Second, high disclosers exhibited *higher* DBP levels across all phases of the experiment, including post-disclosure recovery. It is unclear why a discrepancy between systolic and diastolic blood pressure was found. Third, if the act of disclosure leads to reduced blood pressure, we would expect post-traumatic levels to be lower than post-trivial levels, where little actual disclosure is taking place. Results show, however, that there is virtually no difference in blood pressure following traumatic-event disclosure and trivial-event disclosure, suggesting that "disclosing" a trivial event may be just as healthy as disclosing a traumatic one.

Therefore, while the relationship between repression and hypertension has been strongly supported by research, there is little evidence that repression acts as the causal variable in this relationship. A much larger literature, however, supports the notion that hypertension may actually result in repressive phenomena. Eisenbud (1939) was one of the first researchers to state that hypertension may induce repression. Eisenbud speculated that repression occurred as a consequence of sympathetic nervous activity and was a dependent effect of sympathetic system chemicals on the brain.

Research on animals and humans has indeed supported this hypothesis. As mentioned previously, in the classic psychosomatic position on the repression/hypertension relationship, hypertension is a by-product of repression and that repression is an attempt at avoiding anxiety or negative emotional material. Research in the hypertension literature, however, suggests that the range of cognitive/psychological impairment associated with hypertension extends well beyond emotional matters. Apter, Halstead, and Heimburger (1951) suggested that "impairment of cerebral functions equivalent to that seen in patients with surgical removal of both frontal lobes may occur early in the course of essential hypertension without neurological signs" (p. 812; as cited in Madden and Blumenthal, 1989). A review by Waldstein, Manuck, Ryan, & Muldoon (1991) gathered evidence that ". . . hypertensives are found to perform more poorly than normotensives, particularly on tests of memory, attention, and abstract reasoning, and less consistently on tests of perception, constructional ability, mental flexibility, and psychomotor speed" (p. 451).

Relationships between hypertension and memory have also been described in the literature. Hypertension has been found to be associated with poorer functioning in verbal

memory (Franceschi, Tancredi, Smirne, Mercinelli, & Canal 1982; Mazzucchi, Mutti, Poletti, Ravanetti, Novarini & Parma, 1986; Schmidt, Fazekas, Offenbacher, Lytwyn, Blematl, Niederkorn, Horner, Payer, & Friedl, 1991; Waldstein, Manuck, Ryan, & Muldoon, 1991), visual memory (Franceshi et al., 1982; Mazzucchi et al., 1986; Waldstein et al., 1991; Waldstein, Ryan, Manuck, Parkinson, & Bromet, 1991), and tactile memory (Elias, Robbins, Schultz, Streeten & Elias 1987; Pentz, Elias, Wood, Schultz, & Dineen, 1979). These results are important because they show that inaccessibility of emotional material is not unique in its relationship to hypertension. There is, therefore, no reason to award repression a special place in causing hypertension, as it may be just one of many mental phenomena associated with hypertension.

The causative role of hypertension in cognitive deficiencies involving memory would be further supported by findings that antihypertensive agents reverse or reduce deficiencies previously exhibited by hypertensives. Since untreated hypertensive adults clearly demonstrate impaired cognitive functioning when compared to normotensives, it is possible that these effects might be negated by medicinal treatment of high blood pressure (Muldoon, Waldstein, & Jennings, 1995). Indeed, there is some research that show antihypertensive medication can improve performance on cognitive tasks.

Miller, Shapiro, King, Ginchereau, and Hosutt (1984), in an initial test of 41 matched pairs of hypertensive and normotensive subjects, showed that hypertensives performed less well on a variety of sensory-perceptual tasks, cognitive tests, and psychomotor function tests. Fifteen months later, subjects were retested with the complete battery of 15 tests. Those subjects who had been placed on anti-hypertensive medication (N=21) in the interim period demonstrated improved performance greater

than that of untreated hypertensives (N=13) in 12 of the 15 tests and greater than normotensives (N=24) on 10 of 15 tests. Untreated hypertensives, on the other hand, actually performed more poorly on 7 of 15 tests, while making no or insignificant gains on the other tasks. It is important to note that the only significant changes in blood pressure across the 15 months were exhibited by the treated hypertensive group. The authors conclude that medicinal treatment of hypertension restored cognitive functioning almost to the level of normotensive functioning while untreated hypertensives improved only slightly or actually deteriorated on the tasks. Of course, the above study is not experimental in that administration of antihypertensive medicine was not randomly assigned. Still, the results suggest that blood pressure, as a manipulated variable, can affect chances in cognitive functioning.

Richards, Emsley, Roberts, Murray, Hall, Gao, & Hendrie (2000) investigated the association between hypertensive medication and cognitive deficiencies/dementia in a large (N=2212), African-American sample. They found that antihypertensive (but not antidiabetic, antihyperlipidemic, or antithrombotic) medications were associated with reduced cognitive impairment. The authors suggest that antihypertensive medication may be one way to reduce the cognitive decline usually associated with aging. These results have been criticized, however, due to lack of repeated blood pressure measurements and failure to assess compliance to the antihypertensive treatment (Gambassi, Onder, & Bernabei, 2001).

A review of the neurophysiological effects of antihypertensive medication yielded mixed results (Muldoon, Waldstein, & Jennings, 1995). Given the many types of antihypertensive agents used in studies (beta-blockers, diuretics, calcium channel

blockers, ACE inhibitors, and central sympatholoytic agents), results are difficult to compare. In many cases, the possible beneficial effects of lowered blood pressure may be countered by other activities of the drug. For example, there is some evidence that central sympatholytic agents may have a negative effect on cognitive functioning (Johnson et al., 1990). Unfortunately, the medication used to lower blood pressure is a serious confound, as studies make use of many different drugs, all of which may have either positive or negative effects on performance through channels other than blood pressure. For this reason, and because many studies made use of very small sample sizes, the authors of the meta-analysis conclude that little can be determined concerning the possible effects of hypertensive medication on cognitive performance (Muldoon, Waldstein, & Jennings, 1995).

Stronger evidence for the physiology-first model of repression/hypertension comes from experimental manipulation of blood pressure on pain tolerance. Dworkin et al. (1979) induced high blood pressure in rats with an injection of phenylephrine (a post-synaptic adrenergic agonist) while giving a control group of rats an injection of saline. The high blood pressure group exhibited less escape-avoidance behavior (measured by amount of treadmill running to deactivate electric shock) than the saline group. The authors concluded that this effect was due to the attenuated aversiveness of the noxious stimuli, rather than due to any direct action on the rats' muscular tone or ability to escape.

Zamir and Segal (1979) experimentally elevated rats' blood pressure by applying a solid silver clip on the left renal artery. A control group went through a sham procedure in which no clip was applied. The rise in blood pressure among the rats with blocked renal arteries was found to be closely related to a rise in pain threshold. These findings

support the conclusions offered in the Dworkin et al., (1979) study: blood pressure itself, rather than any particular drug, is the key factor in inducing antinociception (reduction in experience of pain). By raising blood pressure levels, whether by pharmacological or surgical means, increased tolerance to painful stimuli can be produced. The rats' behavior may also be analogous to that exhibited by repressive copers. In both humans and rats, high blood pressure is associated with less experience of pain and/or pain avoidance. However, while the repressive coping literature is correlational, the animal research points to a specific direction: elevated blood pressure causes a reduction in pain experience or pain expression.

Although a strong case for the role of high blood pressure in diminished cognitive functioning and increased pain tolerance can be made, potential biological mechanisms for this relationship need to be identified. In the Dworkin et al. (1979) study, while elevated blood pressure did reduce escape-avoidance behavior, it *did not* reduce that behavior in rats with surgically denervated baroreceptors (receptors specifically designed to detect and maintain blood pressure levels). Furthermore, direct stimulation of the baroreceptors has been found to produce the same behavioral results as raising blood pressure (Randich and Hartunian, 1983; Randich, 1986). Since high blood pressure does not diminish noxiousness when baroreceptors are deactivated, and since direct baroreceptor stimulation does reduce noxiousness, the conclusion may be made that blood pressure operates through activation of the baroreceptors to reduce noxiousness.

Research with naloxone, an opioid antagonist, has further suggested that baroreceptors may play a role in antinociception through their effect on endogenous opioids systems. Saavedra (1981) found that naloxone negated the increased pain

tolerance exhibited by hypertensive rats. Naloxone had no effect on normotensive rats. These results suggest that heightened pain tolerance found in hypertensive rats is somehow related to increased levels of endogenous opiates being produced as a result of stimulated baroreceptors, which are, in turn, activated by elevated blood pressure due to sympathetic arousal.

Human studies have also supported this finding. Janssen and Arntz (2001) injected participants with either naloxone or a saline placebo before having them perform a first-time parachute jump. After the jump, subjects were given electric shocks while subjective measures of pain were recorded. Subjects in the placebo group reported significantly lower pain experience as compared to those who received the opioid antagonist. Furthermore, measures of plasma beta-endorphin levels increased greatly following the jump, further suggesting that the mechanism of pain reduction is through increased opioid production triggered by sympathetic nervous system arousal.

Functionally, endogenous opiates may serve a purpose in diminishing the stress response, through their effects on various emotional and affective systems (Drolet et al., 2001). Stress appears to be the normal stimulus for the production of endogenous opioids (Terman, Shavit, & Lewis, 1984). As such, stress-activated opiates may serve a regulatory/homeostatic role.

The limbic system, in particular, seems heavily affected by the opiate system, as judged by high concentrations of receptors in the amygdala, hippocampus, hypothalamus, cingulate cortex, entorhinal cortex, and septum (Drolet et al., 2001). All of these structures, as part of the limbic system, are important in emotion and memory. Through

effects on these structures, endogenous opiates may regulate the sympathetic system by reducing the stressful response associated with a stressor.

The role of endogenous opiates in attenuating the stress response (including pain and negative affect) could well explain correlational results relating hypertension to repressive coping style. Repression, in the clinical sense of the word, however, also involves memory, such as in the repression of a traumatic incident. Therefore, for the endogenous opiate hypothesis to be a reasonable explanation for the formation of repression, it must also be shown that these neuropeptides have a demonstrable effect on memory.

While no studies on human subjects have been performed, there is sufficient evidence from rat research to suggest that high levels of endorphins, when administered artificially, can serve to block memory processes, specifically through the formation of retrograde amnesia. Beta-endorphin, one endogenous opiate peptide, was shown to cause retrograde amnesia (Izquierdo et al., 1980). Rats were trained to avoid shock in a shuttle avoidance task. On the last trial, rats given saline still showed evidence of learning; exhibited by better performance. Rats given beta-endorphin, however, showed evidence of amnesia as manifested by no improvement over training performance. Beta-endorphins appeared to wipe out all memory of the task. Furthermore, the greater the dose, the greater the amnesic effects.

Introduction of beta-endorphin during learning acquisition has also been found to interfere with long-term memory. Rats given beta-endorphin directly following an inhibitory avoidance task showed no evidence of learning when tests were performed 24 hours later (Castellano et al., 1993). These results suggest that the lack of avoidance

behavior cannot be attributed just to the analgesic effect of endorphins. The effect of beta-endorphin on memory has also been seen in areas other than escape-avoidance, such as in spatial working memory (Wan, Givens, and Olton, 1995), with administrations of endorphin resulting in significantly impaired accuracy in mazes. Naloxone, however, negates the amnesic effects of endorphins, whether those endorphins are introduced artificially (Flood, Garland, & Morley, 1993, Izquierdo et al., 1980) or produced by the organism (e.g., as a response to electroconvulsive shock; Carrasco, Dias, and Izquierdo, 1982).

Therefore, in looking at the literature on repression and hypertension, the following statements can be made: First, there is strong evidence for an inverse relationship between hypertension and diminished reporting of pain, stress, and negative affect. Second, the inverse blood pressure/pain sensitivity relationship can be induced in normotensives with experimentally manipulated, heightened blood pressure. Third, little evidence supports the hypothesis that repression causes increases in blood pressure or the development of hypertension. Fourth, experimentally induced increases in blood pressure and baroreceptor activity in rats has resulted in behaviors similar to those of human psychological repression. Fifth, research on the introduction of opiate antagonists to humans and animals suggests that the antinociceptive effects of high blood pressure are largely due to endogenous opiate activity. Sixth, endorphin-mediated cortical suppression has resulted in memory difficulties similar to those of repression.

The primary function of baroreceptors is the maintenance of proper blood pressure levels. More specifically, they ensure that the brain receives an optimal level of blood across different activities and stressors. Blood pressure levels above optimal could

result in cerebrovascular damage, including stroke. In the case of stress-induced elevations of blood pressure, baroreceptor activity triggers quick recovery of blood pressure levels by acting on cardiovascular functioning via the medulla oblongata. The resulting activity reduces norepinephrine (sympathetic output) and increases acetylcholine (parasympathetic output) levels, thus decreasing blood pressure through reduced heart rate and increased vasodilation. However, limbic and neocorticol influences (i.e., conscious experience of threat) may also necessitate an additional, indirect path for suppressing the sympathetic response. Psychological factors (e.g., distress, fear) contributing to heightened blood pressure may also have to be suppressed in order to regain a homeostatic balance in cardiovascular functioning. The endogenous opioid system may have evolved as the mechanism for inhibiting these psychological influences.

The endogenous opioid system, acting on many limbic structures of the brain, decreases the experience of stress and pain. The net effect of these opiates, when triggered by baroreceptors, may be the diminishing of psychological stress for the purpose of lowering blood pressure levels. An individual experiencing the effect of high levels of opiates would likely experience less pain and stress, and feel less threatened by psychological stressors, consequentially reporting less pain, less stress, and less negative affect. These signs, together with heightened blood pressure levels, are the exact set of behaviors that repressive copers present in laboratory studies. As suggested by others (see Nyklicek, Vingerhoets & Van Heck, 1998) the repressive coping style, as a psychological phenomenon, may be the result of a physiological state, mainly, elevated blood pressure, baroreceptor activity, and endogenous opioid levels.

While the literature is suggestive of a causal role of hypertension in repressive phenomena, this hypothesis has never been specifically tested. In order to more fully understand the relationship between the two conditions, previous research must be extended beyond physical pain, to include concepts more cognitive in nature. The present experiment is designed to test the hypothesis that endogenous opioids mediate the relationship between hypertension and repression. If endogenous opioids are the mechanism in this relationship, opioid antagonism should decrease repression and increase disclosure.

In the present study, normotensives and hypertensives participated in four laboratory sessions in which they verbally disclosed their current concerns, stressors, and feelings. Following the narratives, participants filled out scales measuring levels of repression and disclosure. These measures included a measure of life concerns and an index of repressive coping (The Index of Self-Regulation of Emotion; Mendolia, 2002) which is a composite of social desirability and manifest anxiety scales. In two of the sessions, individuals were administered naltrexone, an opioid antagonist. In the other two sessions, a placebo was administered. Administration was double-blind and the schedule was randomized.

Planned comparisons were used to test the hypothesis that naltrexone would significantly alter the self-reports of hypertensives, but not normotensives, in the direction of less repression and greater disclosure. In planned comparisons, real differences among means are more likely to be detected (Glasnopp & Poggio, 1985). These tests are more powerful than *post hoc* tests and are thus recommended when comparing means (Pedhazur, 1982). Furthermore, planned comparisons reduce spurious

findings by reducing the total number of tests and minimizing the impact of chance (Thompson, 1988). As low subject numbers would prevent sufficient power to run initial interaction analyses, the effects of naltrexone were statistically tested separately on each the normotensive and hypertensive groups. If naltrexone was found to significantly affect the dependent variable of repression/disclosure on hypertensives, but not normotensives, this finding would lend support for the role of endogenous opioids in mediating the hypertension/repression relationship.

In addition to the two main dependent variables (life concern disclosure and repressive coping), a number of measures were included to test discriminant validity and measure potential confounds. If naltrexone was found to increase disclosure of negative events and experiences, it might be argued that the change was due purely to decreased mood. If individuals experienced physical symptoms such as fatigue or nausea, they may feel worse and thus report more negative experiences. Because naltrexone has been reported by some early investigators as negatively affecting mood (Mendelson, Ellingboe, Keuhnle, & Mello, 1979; Hollister, Johnson, Boukhabza, & Gillespie, 1981), a measure of mood states was included in the present study to study the possible confounding effects of mood. The Profile of Mood States (POMS; McNair, Larr, & Dropplemen, 1971), used in the present study, is a standard measure of mood in similar investigations.

As endogenous opioid antagonism is known to affect perception of physiological pain (e.g., Janssen and Arntz, 2001), a test of pain was provided by the inflation of a blood pressure cuff prior, during, and following the interview period. Although the pain evoked by the readings is usually minimal, it is possible that experienced pain would

increase with opioid antagonism. If pain perception is increased, it might be argued, similarly to mood, that increased reporting of negative affect was due to increased physiological pain. Following the last blood pressure reading, participants were asked to rate the pain experienced from the cuff. These ratings were used to determine if experienced pain increased with opioid antagonism and if physiological pain could potentially confound results obtained with the main dependent variables of repression and disclosure.

Alexithymia is a condition defined by difficulty in recognizing emotional states, problems with articulating emotions, a highly mechanical, analytical, and externally oriented thinking style, and possibly an impoverished fantasy life (Sifneos, 1996; Parker, Bagby, Taylor, Endler, & Schmitz, 1993). Although repressive coping and alexithymia are thought to be independent phenomena, they share some common traits, such as inability to express emotion (although the repressive coper may selectively inhibit the expression of dystonic emotions). As the cognitive-affective disturbances evidenced by alexithymics (see Taylor, 1984) are likely different in origin than than those evidenced by repressive copers, a scale of alexithymia (Taylor, Ryan, & Bagby, 1985) was included in the present design to provide discriminant validity. Endogenous opioid antagonism, and thus the drug naltrexone, should have no effect on alexithymic symptoms.

Finally, conscious knowledge of the drug condition could affect material provided in interviews and responses to self-report measures. If participants knew when they received the active drug, they might behave as they expect they should under such a drug. While participants were not told of any possible cognitive and emotional effects, they were informed of the drug's effect on analgesic systems. To serve as a manipulation

check, subjects were asked to guess, at the end of each session, whether they received the drug or placebo. If participants could correctly guess which substance they had received, their behaviors might be influenced by such knowledge.

While no cardiovascular effects of naltrexone have been reported, the effects of the drug on systolic and diastolic pressure where measured. If high blood pressure is maintained in order to hold endogenous opioid activity at a certain level, then antagonism of those opioids might drive systolic blood pressure higher in an effort to restore equilibrium. To the extent that blood pressure is responsible for the maintenance of opioid levels, blood pressure should rise, by way of compensation, whenever those opioid levels fall. Therefore, we would expect blood pressure levels to be higher among individuals under the drug condition. If the blood pressure-opioid-repression condition holds only for hypertensives, however, we would not expect such a change in normotensives. While changes in systolic and diastolic blood pressures are affected by different mechanisms (cardiac output and vasodilation, respectively), both may increase under acute stress. Because systolic blood pressure has been found to be more reactive during social tasks (such as giving a personal interview), it might be expected that systolic pressure would be more strongly affected than diastolic in the present study.

CHAPTER II: METHOD

Participants

Male, undergraduate students were solicited via extra credit announcements for the Department of Psychology at the University of Tennessee. Volunteers were invited to participate in a short cardiovascular/medical screening procedure for nominal extra credit in a psychology course. The screening sample consisted of 125 individuals.

After signing the consent from, participants completed a blood pressure screening procedure. Readings were taken with a Critikon Dinamap 1846 Vital Signs Monitor at 2-minute intervals for a 15-minute period. Resting levels were calculated by averaging the last 3 readings. Volunteers were considered potential participants in the experiment if their systolic blood pressure fell in the upper or lower 10^{th} of the screening distribution.

Following the cardiovascular screening period, participants were given a short medical history questionnaire. The questionnaire screened for two types of conditions: 1) secondary causes of hypertension and 2) conditions contraindicated with the administration of naltrexone. Because the most common causes of hypertension in young adults are renal disease and diabetes, causes of secondary hypertension focused on signs and symptoms of these disorders. Those reporting diabetes or kidney problems were also excluded from further participation. Furthermore, those reporting symptoms common in renal disease (jaundice, chronic fatigue, chronic nausea, chronic fever, "foamy" urine, difficulty urinating, painful urination, blood in urine, or swelling in the hands, feet or around the eyes) were excluded from the study.

A number of conditions considered dangerous with administration of naltrexone were also screened out. These conditions and situations include: alcoholism, narcotic

abuse, use of narcotic medication, and hepatitis. Furthermore, although naltrexone has not been found to have a significant impact on mood (Hatsukami, Mitchell, Morley, Morgan, & Levine, 1986; Malcolm, O'Neil, Von, & Dickerson, 1987; Miotto, McCann, Basch, Rawson, & Ling, 2002), participants were screened for depression, dysphoria, or a history of mental disorders. Lastly, participants were instructed to list all current medications, and those taking medication contraindicated with the use of naltrexone (primarily narcotic analgesics) were screened out from further participation. Participants indicated on a form whether or not they would be interested in continuing with the study, should they be selected. Five individuals who met cardiovascular criteria were screened out for one of the above reasons. Two reported histories of significant psychological distress, one reported excessive medication use, one reported excessive alcohol consumption and one reported an excessive number of allergies.

From those individuals who were not screened out for medical reasons, 12 were selected for the normotensive group and 12 for the hypertensive group. These individuals were contacted, given a brief overview of the study and invited to return to the lab for a second assessment of their blood pressure status and further information on the study. Individuals selected for the study were paid $50 for their participation. Twenty-two individuals, eleven in each the hypertensive and normotensive groups, accepted the invitation to participate in the experiment. Of these individuals, two withdrew before their first session, citing concern (or parental concern) over the use of drugs in the experiment. Both of these individuals were in the normotensive group. One further individual was terminated by the experimenters due to a missed first session. This individual was deemed to be an unreliable participant for the experiment.

The remaining sample, all male, ranged in age from 18 to 25 years (mean 20.28 years). The participants were all Caucasian, with the exception of one African-American, who was in the hypertensive group.

Measures

The abbreviated version of the Profile of Mood States (POMS; McNair, Larr, & Dropplemen, 1971) contains 30 positive and negative emotional states (such as tense, lively, sluggish, and angry) to which the participant indicates to what degree they have felt that way in the past week. Responses are made on a Likert-type, 5-point scale. The subscales of the abbreviated version has yielded alphas ranging from .66 to .95, with a mean of .80, indicating good internal consistency.

Disclosure of life concerns was measured by the Life Concerns Checklist (LCC; Rayner & Price, 1989). The LCC contains 34 areas in which a person may be concerned about in his life. Examples of items are: "conflict with loved ones," and "feeling inadequate as a person." Respondents indicate, on a 3-point scale, whether they are *never* concerned, *somewhat* concerned, or *very* concerned about each area. The scale has good test-retest reliability over 5-weeks ($r = .85$, $p < .01$). Internal reliability, measured with the Kuder-Richardson formula for scales with unidimensional constructs, was high (rKK = .93).

The most widely used method of measuring repressive coping style is to cross the scores of two scales: the Taylor Manifest Anxiety Scale, Bendig Short Form (MAS; Bendig, 1956) and the Social Desirability Scale (MCSDS; Crowne & Marlowe, 1960). The MAS contains 20 items measuring anxiety, such as, "I often find myself worrying about something" and "I am more self-conscious that most people." Individuals respond

to each statement with true or false. The MCSDS contains 33 items that measure how defensive one is about their own behavior or thoughts. Examples include, "I never resent being asked to return a favor," and "I have never deliberately said something that hurt someone's feelings." Respondents indicate whether the statement is true or false in describing them. Repression was measured with the most recent method of subtracting the MCSDS score from the TAS, which yields the Index of Self-Regulation of Emotion (ISE; Mendolia, 2002). This method provides a continuous measure of the MCSDS/TAS relationship. High-anxious individuals have lower scores on the spread while repressors occupy the higher end of the index. Low-anxious and defensive high-anxious make up the middle portion of the spread.

Alexithymia was measured with the Toronto Alexithymia Scale, 20-item version (TAS; Taylor, Ryan, & Bagby, 1985). The TAS contains items such as, "I have feelings that I can't quite identify" and "I prefer to analyze problems rather than just describe them." Respondents reply on a 5-point, Likert-type scale. Endorsement of the items suggests high alexithymic traits. The 20-item version of the TAS has been found to have high internal consistency ($\alpha = .85$)

As endogenous opioids are known primarily for their antinociceptive/analgesic properties, most experimental research has been conducted on the effect of opioid antagonists on pain. As previous research has found opioid antagonists to increase experienced pain in hypertensives but not normotensives, blood pressure cuff pain was measured in the present study as an attempt at replication. Blood pressure measurements were taken at 2-minute intervals for 5 minutes prior to each interview (baseline). These measurements continued for the duration of the interview (task) and for 2 minutes

following the interview (recovery). Because inflation of the cuff necessitates the occlusion of the blood vessels, a certain amount of ischemic discomfort may be expected. Most individuals typically find this discomfort to be minimal, however, some find the pressure painful. Following each session, the participants were asked to rate, from 1 to 10, how painful they found the blood pressure cuff inflation for the duration of the measurement process.

Materials

Experimental manipulation of opioid activity was achieved with the opioid antagonist naltrexone, which is frequently used in treatment of narcotic dependency (Malcolm, O'Neil, Von, & Dickerson, 1987). Naltrexone effectively blocks activity at *mu* and *kappa* opioid receptors (Preston & Bigelow, 1992). It is orally absorbed, reaches peak absorption levels in an hour, and has a half-life of approximately 4 hours (Martin, Straughn, Lo, Schary, & Whitney, 1984). In healthy volunteers, naltrexone has not been shown to present any significant adverse effects (Martin, Jasinski, & Mansky, 1973).

Revia brand naltrexone is administered as a 50mg tablet. For the present experiment, these tablets were inserted into a green opaque gelcap, in order to maintain similarity in appearance with the placebo. An equal amount of nonactive substance (sugar pill) was placed in a gelcap to serve as the placebo.

Procedures

Participants who met blood pressure requirements on the two assessment periods were asked to schedule four lab visits over a two-week period, with each visit lasting approximately 1 hour and 45 minutes. Upon arrival, participants were administered either

the naltrexone or placebo, in a double-blind, randomized fashion. Each participant was given the active drug on two occasions and the placebo on two occasions.

Following drug or placebo administration, participants were seated and their cardiovascular group status (high or low) was reconfirmed. Following confirmation of group status, participants waited for 1 hour to allow for peak absorption of the drug. During this time, participants were allowed to bring their own work or they could choose between pre-screened television programs, video games, and magazines. Available media were screened to insure the absence of strong emotional content that could confound later data collection of affective states.

Following the one-hour absorption period, blood pressure was again checked. Blood pressure state was assessed with three readings taken at two minute intervals. These readings both established a baseline level for pre-interview cardiovascular status and allowed tests to be conducted on the cardiovascular effects of the opioid antagonist.

Following baseline cardiovascular measurements, participants completed the interview stage. These interviews were audiotaped for later transcription and analysis. Blood pressure measurements continued at two-minute intervals throughout the duration of the interview. Participants were asked three questions, which were not counterbalanced for order by session. Following each question, participants were given 20 seconds to think of a response and were then allowed to describe their response. Participants were first instructed to, "Let your mind wander for a short period. After about twenty seconds, you can describe to me what you were thinking about." Secondly, participants were instructed to, "Think for a few moments about the past 24 hours. Review the events, good, bad, and neutral, in your mind. In a few moments, I will have

you describe those events and generally how the last 24 hours went in as much detail as you would like." Finally, participants were asked, "What is the one thing that is causing you the most stress, anxiety, or concern right now? Think about that for a few moments and then you can describe that situation to me." Participants were allowed to respond to the questions without interruption and no followup questions were asked. This open-ended approach allowed the coding of spontaneous disclosure (both length and depth) and minimized confounding by interviewer biases.

Following the narrative, participants filled out the scales of repression/disclosure: POMS, LCC, positive and negative valenced descriptors, TAS, and MCSDS. Two additional blood pressure measurements were taken during this period to serve as a pseudo-recovery period.

CHAPTER III: RESULTS

Early Termination

Of the individuals who attended the first session, only one terminated before completion of the 4-session protocol. The participant, in the hypertensive group, withdrew from the experiment after the first session. In the first session, baseline readings averaged 135/72 mmHg. During the interview, the subject talked very little, but exhibited large blood pressure increases in reaction to talking about a test which was imminent. Blood pressure levels averaged 157/115 mmHg during the interview period. The participant withdrew from the study the day after the first session, citing negative side-effects of the drug. Specifically, he mentioned "getting into fights" with people on the internet, and "feeling his blood pressure rise." He attributed these experiences to the drug. He also tried playing basketball the evening of the first session but was unable to continue due to fatigue. The participant went to bed early and felt fine the next day. After officially terminating the participant, it was confirmed that he did receive the active drug on the first session. The individual was paid $5 for his participation in the first session.

Group Differences

Blood pressure levels were measured on three separate dates to insure accuracy of hypertensive or normotensive status. The first date was in the initial cardiovascular/medical screening which determined group status. Independent t-tests indicated a clear distinction in both systolic (SBP; $t(16) = 18.0$, $p < .0001$) and diastolic (DBP; $t(16) = 3.9$, $p < .0001$) blood pressure.

After a minimum of one week after the initial screening, participants returned to the lab for blood pressure confirmation. Figure A-1 (all figures and tables are located in

the Appendix) presents the groups differences in SBP and DBP. As can be seen in the boxplots, there was no cardiovascular overlap between the groups when reassessed one week later. Independent t-tests confirm that SBP ($t(16) = 13.8$, $p < .0001$) and DBP ($t(16) = 6.2$, $p < .0001$) remained significantly different between groups. The cardiovascular means for each participant are presented in Table A-1. Reported means were calculated by averaging the last 3 readings taken during the screening (a) and confirmation (b) sessions.

As body mass is a known factor in elevated blood pressure, the body mass index (BMI) was calculated for each subject. BMI is a better measure of obesity than weight alone and is calculated with the following formula: (weight [lbs] x 704.5) / (height [in] x height). BMI was significantly different between groups ($t(16) = -.37$, $p = .002$) with hypertensives having a higher BMI (mean = 24.9, sd = 1.8) than normotensives (mean = 22.0, sd = 1.4). Therefore, in all tests involving cardiovascular dependent measures, BMI was included as a covariate.

Groups did not differ on age (t(16) = -.5 (p=.617). No other variables were assessed.

Main Analyses: Effects of Naltrexone Administration

As the sample size was too small to test for interaction effects, simple effects for drug/placebo were tested separately for the normotensive and hypertensive groups, according to *a priori* hypotheses. Custom contrasts were used to compare the drug and placebo sessions.

Confounds and controls

Before analyses on main dependent variables were conducted, potential confounding variables were tested. These variables included pain, mood, and participants' guess of drug status.

Pain ratings, provided by the participants following the last blood pressure reading of each session was tested as a function of drug/placebo condition. No significant drug/placebo effect was found for either hypertensives ($F(1) = 0.00$ ($p = 1.00$) or normotensives ($F(1) = 3.769$ ($p = .110$). Therefore, both groups failed to respond differentially to drug/placebo conditions. Most likely, blood cuff pain served as a poor measure of pain sensitivity, as the pain was not constant (as with a dedicated cold-pressor or finger-pressor procedure) and most individuals do not find the inflation to be uncomfortable.

Consistent with the most resent research on naltrexone and mood, there was generally little effect for drug condition on mood, as measured by the Profile of Mood States. The anger, confusion, fatigue, depression and vigor subscales were unrelated to drug condition in either group. The tension subscale was significantly related to drug condition in normotensives ($F(1) = 20.86$, $p = .003$) but not in hypertensives ($F(1) = .018$, $p = .90$). As seen in Figure A-2, normotensives reported significantly greater tension during the drug condition while hypertensives remained unchanged.

Chi-square analyses were used to determine whether or not participants could accurately assess whether they had received the drug or placebo. Neither hypertensives ($\chi = .48$, $p = .59$) nor normotensives ($\chi = 1.8$, $p = .22$) guessed their drug or placebo condition over chance accuracy.

Further control was provided with the Toronto Alexithymia Scale, as the condition is thought to have origins distinct from repressive coping, despite some superficial similarities between the two conditions. There was no significant effect for the drug condition on alexithymic traits in hypertensives ($F(1) = .05$, $p = .82$) or normotensives ($F(1) = 3.04$, $p = .12$). Participants experienced no change in alexithymic symptoms as a result of opioid antagonism.

Drug effects on repression/disclosure

Contrasts revealed a significant main effect for the drug condition on hypertensive's reporting of life concerns ($F(1) = 5.07$, $p = .05$). As seen in Figure A-3, hypertensives disclosed significantly greater life concerns when under the active naltrexone sessions. Life concerns reported by normotensives did not change as a function of drug/placebo ($F(1) = .01$, $p = .944$).

As measured by the Index of Self-Regulation of Emotion, hypertensives exhibited significantly less repressive coping in the drug condition ($F(1) = 20.83$, $p = .004$). Normotensives, however, showed no significant change ($F(1) = .73$, $p = .427$). Figure A-4 presents the differential responding of hypertensives and normotensives to the drug condition. Although it appears that normotensives in the drug condition display a *greater* tendency towards repressive coping, it is important to note that this difference is not significant.

Cardiovascular effects

As no cardiovascular effects for opioid antagonism have been reported in previous studies, the potential effects of naltrexone on blood pressure was investigated in the present study. If hypertension is related to endogenous opioid systems, the

manipulation of one system might have a demonstrable effect on the other. Blood pressure measurements were taken at 2-minute intervals for six minutes prior to each interview (baseline), during the interview (task), and for 2-minutes after the interview (recovery).

Systolic blood pressure was compared for high and low pressure groups across drug conditions in the baseline, task, and recovery phases. The drug condition had a significant effect on systolic blood pressure during interview ($F(1) = 24.14$ (p=.001) and recovery ($F(1) = 7.50$ ($p = .02$) for the hypertensive group but not for the normotensive group ($F(1) = .0$, p=1.0; $F(1) = .62$, $p = .49$). Hypertensives exhibited significantly higher systolic blood pressure levels during the interview when in the drug condition as compared to the placebo condition. For normotensives, there was no significant difference in levels during the interview. Figure A-5 presents systolic levels over the three phases for hypertensive and normotensives in the drug and placebo conditions. As can be seen in the graph, normotensives exhibited the same response whether under the drug or placebo. In hypertensives, however, the drug condition produced significantly higher systolic levels under the interview. No effects were found for drug on diastolic blood pressure.

Narrative effects

Total number of words contained in the participant narratives were counted. A general contrast found a significant main effect for drug condition ($F(1)$=7.21, p=.02). As seen in Figure A-6, both the hypertensive and normotensive groups were similarly affected by the drug condition. Both groups provided more words in their narratives

under the drug condition, supporting the hypothesis that the drug condition increases disclosure.

CHAPTER IV: DISCUSSION

A good body of literature has reported a positive relationship between hypertension and diminished reporting of stress and negative affect (e.g., Cumes, 1983; Handkins & Munz, 1978; Winkleby, Ragland & Syme, 1988; Davies, 1970; Rose, Jenkins & Hurst, 1978). Very few studies, however, have attempted to discover the nature or mechanisms of this relationship. While many clinicians and theoreticians have hypothesized a model in which inhibited affect manifests itself somatically (e.g., Esterling, Antoni, Kumar & Schneiderman, 1990; Shedler, Mayman, and Manis, 1993), little evidence has been brought forth to support this claim. Likewise, while studies have found manipulation of physiological factors (such as blood pressure and endogenous opioid levels) to affect *pain* perception (e.g., Jannsen & Arntz, 2001), no attempts to extend this model to repressive phenomena have been made. The present study presents perhaps the first attempt to test the model that altered physiology, specifically the levels of endogenous opioids controlled by blood pressure, can affect the manifestation of repressive coping phenomena.

Eighteen participants, 10 hypertensive and 8 normotensive, were administered naltrexone, an opioid antagonist, or a placebo across four laboratory sessions according to a randomized and double-blind schedule. In each session, measures of disclosure and repressive coping were collected. Overall, the drug condition was found to reduce repressive phenomena and increase disclosure *in hypertensives only*, supporting the hypothesis that cardiovascular-controlled opioids are, at least partially, responsible for the diminished responding evidenced by hypertensives.

Summary of Findings

Controls and potential confounds

Discriminant analyses and tests of potential confounds were performed on four variables: pain, mood, alexithymia, and the manipulation check (participants guess of drug/placebo condition).

First, increased experienced physiological pain and negative mood are two areas in which opioid antagonism may have an effect. Since increased pain and a more negative mood could influence responses to narrative questions and self-report scales, these variables were included in the study design. Neither the hypertensive nor normotensive group experienced greater pain as a function of the drug condition. Levels of pain reported from the blood pressure cuff were similar across drug/placebo conditions. Therefore, it is unlikely that increased physiological pain was responsible for increased disclosure.

The Profile of Mood States is a popular measure of general mood with subscales for tension, depression, anger, fatigue, vigor, and confusion. While a small number of studies have found a negative effect of naltrexone on mood (Mendelson, Ellingboe, Keuhnle, & Mello, 1979; Hollister, Johnson, Boukhabza, & Gillespie, 1981), these studies have been criticized for small sample sizes and lack of appropriate controls (Malcolm, O'Neil, Von, & Dickerson, 1987). Recent studies have failed to find any dysphoric effects of naltrexone (Hatsukami, Mitchell, Morley, Morgan, & Levine, 1986; Malcolm, O'Neil, Von, & Dickerson, 1987) and a review of the literature found no evidence for a significant effect on mood (Miotto, McCann, Basch, Rawson, & Ling, 2002). If naltrexone significantly alters mood, those mood changes may be responsible

for effects observed in the repressive scales. In other words, perhaps naltrexone just makes people feel bad and this is why there is more observed disclosure. The results of the present study do not support this hypothesis of a possible confound, however. Five of the six subscales were unrelated to drug condition in either the hypertensive or normotensive groups. The one subscale with significant differences, tension, was significant only in the normotensive group. Therefore, significant results for disclosure observed in the hypertensive group are unlikely to be caused by alterations in mood.

Participant's knowledge of their drug/placebo condition may influence their responses to interview and scale questions. Results show that participants fared no better than chance at guessing whether they received the drug or placebo. Therefore, psychological differences were found (decreased repression and increased disclosure) despite no consciously-experienced, internal change in the participants. This finding is important for two reasons; first, it adds strength to the repression/disclosure findings by suggesting they are not simply the result of somatically-experienced changes. Second, it shows that the effects of endogenous opioids may be stronger on repression and disclosure than on the systems (pain, mood) with which opioids are traditionally linked. The results from the pain, mood, and guess measures suggest that none of these factors were responsible for psychological differences found between placebo and drug conditions.

Finally, although alexithymia and repressive coping are observed to share common traits, such as impairments in emotion recognition (Lane, Sechrest, Riedel, Shapiro, & Kasniak, 2000), evidence suggests that these two phenomena are independent (Newton & Contrada, 1994). In fact, despite their superficial similarity, repressive copers

score extremely low on scales of alexithymia (Myers, 1995). Since alexithymia is also found in greater proportions among hypertensives (Todarello, Taylor, Parker, & Fanelli, 1995), a scale of alexithymia was included in the present study to assess whether alexithymia traits might be similarly affected by opioid manipulation. If alexithymia is independent from repressive coping and involves different physiological and neurological conditions in its genesis and maintenance, opioid antagonism should have no effect on alexithymia scores. Indeed, neither the hypertensive nor normotensive group were found to differ on alexithymia scores as a function of drug condition, therefore, it was concluded that alexithymia does not share physiological aspects with repressive coping. These findings support the general finding that alexithymia and repression are separate constructs. Furthermore, it supports the hypothesis that opioid antagonism has specific effects on certain psychological phenomena (repression and disclosure) while not affecting other similar, but independent, constructs.

Repression and disclosure

While adequate power existed for the within-subjects drug variable, there was insufficient power to conduct tests of group differences (normotensive versus hypertensive), due to small sample sizes. Therefore, *a priori* comparisons were developed. Specifically, it was hypothesized that the drug condition would have a significant effect on hypertensive disclosure but not normotensive disclosure. The obtained results support these hypotheses. While analyzing groups separately for drug effects does not determine whether slopes are different for the two groups, it does provide a basis for saying the drug condition affects one group but not the other.

Two measures were included to test the effects of the drug naltrexone on disclosure. The Index of Self-Regulation of Emotion is a composite score of the Marlow-Crowne Social Desirability Scale and Taylor's Manifest Anxiety Scale which provides a measure of repressive coping. High repressive copers are those individuals who report low levels of anxiety but also present high levels of social desirability. Planned comparisons showed that naltrexone significantly decreased repressive coping in hypertensives but not in normotensives. Therefore, the repressive coping demonstrated by hypertensives appears to be significantly altered by opioid antagonism. As repressive coping is linked with an impaired ability to recall negative events and material (Myers & Brewin, 1995; Myers, Brewin & Power, 1998) and overly positive evaluations of the self (Myers & Brewin, 1996), the presence of repressive coping may be an obstacle to therapy. Research has long found that repressive copers claim low levels of anxiety, despite physiological indices that indicate repressive copers are more reactive to stress than any other group (Weinberger, Schwartz & Davidson, 1979).

Previous research has also found that hypertensives report fewer life concerns (Rayner & Price, 1989) than normotensives. Again, the unrealistic reporting of concerns and problems would pose potential problems for both personal relationships and psychotherapeutic ones. In the present study, the Life Concerns Checklist was used as a measure of self-report of concerns. Under the drug condition, hypertensives reported significantly more life concerns. Normotensives exhibited no significant changes in life concerns. Thus, hypertensives exhibited a differential reaction to opioid antagonism as compared to placebo whereas normotensives did not.

Narrative effects

Because repressive coping is a condition that likely reduces disclosure in therapeutic settings, participants completed a short interview at each visit. Participants were asked to free associate and describe what they thought about, describe the last 24 hours, and describe their most pressing concern or stressor. In this way, the usefulness of naltrexone as an acute tool in therapy could be assessed. Crude analyses of provided narratives partially supports the hypothesis that opioid manipulation alters verbal communication. Both hypertensives and normotensives spoke significantly more in the drug condition. Across hypertensives and normotensives, individuals were observed to speak an average of 20% more in the drug condition. These results warrant the further investigation of opioid processes on the quality of oral narratives.

Cardiovascular effects

Of particular interest were the cardiovascular effects of the drug condition. Hypertensives under the stress of the interview exhibited significantly higher systolic blood pressure levels with administration of naltrexone than with administration of placebo. These effects were only seen with systolic blood pressure, only in the interview condition, and only with hypertensives. These results would be predicted by the opioid-peptide theory of hypertension/repression. As stated earlier in the introduction, individuals with hypertension may have chronically elevated levels of endogenous opioids. The activity of these opioids on the limbic system may produce the collection of phenomena generally known as repressive coping. The net result of this opioid activity would be to diminish the perception of negative stimuli. Therefore, individuals with chronically elevated levels of opioids would experience negative stress and affect less

than their normotensive counterparts. An acute stressor (such as a disclosing interview) would produce a large sympathetic opioid response which would diminish experienced stress and thus minimize the cognitive experience of threat that would further increase blood pressure. In susceptible individuals, however, the inhibition of this system (as with opioid antagonism) would remove the primary coping strategy used by these individuals. Therefore, negative emotional experience would not be attenuated and increased stress would drive blood pressure even higher. More directly, hypertensive-repressors who rely strongly on endogenous opioids for coping would find events more stressful if those systems were removed. This increased stress would in turn invoke higher systolic blood pressure levels. Figure A-7 graphically represents the theoretical stress-opioid feedback system under normal circumstances and opioid antagonism. Endogenous opioids act as a moderator between objective stressors and subjective stress by attenuating experienced distress. Inhibition of these opioid systems, then, would increase experienced stress and lead to the elevated blood pressure levels observed in the present study.

Implications

Relationship deficiencies in the hypertensive individual

A small literature suggests that hypertensives may possess an altered basis of negative stimulus appraisal (Nyklicek, Vingerhoets, & Van Heck, 1996; Winkleby, Ragland, & Syme, 1988). In particular, hypertensives seem less able to detect negative emotive cues in others (Sapria et al., 1971). If hypertensives are less able to detect anger, hostility, or other negative affective states in others, their ability to adjust behaviors to relieve tension may be reduced. Hypertensives, therefore, may not see serious circumstances for what they are and may minimize the impact of their actions on

relationships. These hypotheses have been supported by previous literature on essential hypertension and familial interactions. Hafner and colleagues (1983), measured the marital quality of twenty-five men and twenty-six women with essential hypertension. Spouses of hypertensive males reported significantly higher marital dissatisfaction than spouses of normotensive males. Similarly, there was a lack of congruence between spouses' scores with the hypertensive females. The authors concluded that anecdotal reports connecting hypertension with marital discord and communication problems were supported.

In another study, the family dynamics were examined in families with both normotensive and hypertensive fathers. In families with hypertensive fathers, gaze aversion during negative verbalizations occurred significantly more than in families with normotensive fathers (Baer et al., 1983). In a second study, families with hypertensive fathers were characterized by less direct coping and an avoidance of conflict resolution. The authors concluded that conflict styles modeled by the hypertensive father may have adverse effects on the family and, more specifically, on the child.

Hypertension, often considered "symptomless" condition, may have important and negative psychological and relationship consequences. Ironically, one of the symptoms of hypertension may be the inability to notice or the minimization of other symptoms of the disease. A hypertensive individual may not only feel fine, but also have a condition that, when removed, makes them feel worse (as the curing of an opioid-addict would be painful to the patient). Under these circumstances, patients may be resistant to the treatment of hypertension, perhaps contributing to the prevalence of the condition.

Implications for psychotherapy

The results of the present study bear important implications for psychotherapy, particularly in the treatment of hypertension, repression, or co-morbid instances of the two. The first important implication is that hypertensives may be particularly difficult patients to treat in a psychotherapeutic environment. Many hypertensives would be unable, or unwilling, to access negative material for presentation in a clinical session. Previous studies have shown that hypertensives are less likely to disclose personal information in both interview and questionnaire format (Berglund, Ander, Lindstrom & Tibblin, 1975; Weiner, Singer & Resier, 1962; Handkins & Munz, 1987; Cumes, 1983; Cumer-Rayner & Price, 1981). This inability or unwillingness to disclose personal information has been cited as a factor which can potentially render an individual unsuitable for psychotherapy (Shands, 1977). Therefore, hypertensive individuals may make exceptionably difficult patients, especially for insight-oriented therapies.

Second, case studies (e.g., Mann & Delon, 1995) have shown a direct correlation between disclosure of traumatic material and large drops in sustained hypertension. Temporal resolution of these studies, however, were insufficient to determine which factor, the disclosure or the blood pressure drop, came first. While the classic interpretation of such results is that the disclosure brought about a change in blood pressure, recent evidence, including the present study, suggest that the converse might be more accurate. If repressive phenomena are driven by cardiovascular-regulated endogenous opioid levels, large drops in blood pressure may allow "repressed" material to surface. Also, if the symptoms of repressive coping are maintained primarily by physiological mechanisms, attempts at controlling hypertension via psychotherapeutic

techniques would be ineffectual. Therefore, psychotherapeutic techniques may not be an effective route to reducing hypertension. Furthermore, in cases of comorbid repression/hypertension, the hypertension may have to be addressed before meaningful psychotherapy can occur.

Third, as the effects seen in the present study apply across the normotensive range, the therapeutic environment, to the extent that it is a stressful one, could also cause the exact physiological changes that facilitate the repression or attenuation of negative affect. The normal sympathetic response to a stressful situation may be an obstacle to therapeutic progress. While efforts to maintain a stress-free therapeutic environment would lessen the impact of stress-induced opioid activity, there is little that could be done to prevent acute stress brought about by discussion of a personal trauma. In essence, as one gets closer to stressful material, the natural sympathetic response and subsequent opioid activity could impede further progress. Ironically, this phenomenon would present itself precisely when the client would need to be least repressive. While the exact manifestations of this response are unknown, it could involve forgetfulness, minimization of the new material, confusion, or perhaps laughter. This phenomenon may manifest itself in the same way as a classically interpreted psychological defense, but its successful navigation may lie more in controlling the sympathetic response than in examining the unconscious motives behind the defense.

The present study, while far from sufficient to recommend naltrexone as an aide to psychotherapy, certainly warrants the further investigation of opioid antagonism in the service of therapy. Certainly, many difficulties are present; at least one-hour absorption time would be required, and this problem alone might make its use impractical for

general clinic use. Also, the potential negative side-effects of the drug (general malaise) which might be exaggerated in the hypertensive-repressor, might reduce adherence to the drug schedule or to the therapy in general. Clients would not be able to consume alcohol for some time afterwards and some chronic pain conditions may worsen. Before acute administration of naltrexone can be recommended, therefore, careful study of its pros and cons in the therapeutic session would have to be carried out.

The development of hypertension

In the United States alone, more than 50 million people are estimated to have chronically high blood pressure levels. Thirty-eight percent of blacks and twenty-nine percent of whites have the condition (Merck Manual, 1997). Hypertension, as the leading cause of stroke and a major cause of cardiovascular disease, is one of the most dangerous conditions in the industrialized world. The causes of essential hypertension, which makes up about 90% of hypertensive cases, are unknown. While several risk factors (e.g., family history, obesity) are known, these factors alone are insufficient to explain the development of hypertension.

The opioid-peptide theory holds that hypertension may be a learned response to stressful stimuli (Dworkin et al., 1979). If increased blood pressure levels can produce antinociceptive effects through baroreceptor-medicated, endogenous opioid activity, heightened blood pressure levels can possibly become a conditioned response. Essentially, elevations in blood pressure provide psychophysiological relief from stressful situations, thus serving as a negative reinforcer for high blood pressure responses (Randich & Maixner, 1984). Some individuals may be particularly susceptible to this response, either because of a hypersensitive cardiovascular system (larger blood pressure

responses) or hypersensitive antinociceptive/analgesic systems (Droste et al., 1994). Furthermore, frequent exposure to stressors (i.e., highly stressful environments) may more often trigger blood pressure and antinociceptive responses. Repeated experiences with this cardiovascular-baroreceptor-opioid system may result in an instrumentally-conditioned, heightened blood pressure response to stressors. Over time, continual reinforcement of elevated blood pressure levels may result in sustained hypertensive conditions. In other words, hypertension may be an operantly conditioned response to repeated exposure to stressors. This hypothesis of learned hypertension is supported by research showing that heart rate and blood pressure can be operantly conditioned in nonhuman primates (Engel & Joseph, 1982; Mitchell, Graham, & Castracane, 1982).

Such a model would have important implications for the prevention and treatment of hypertension. While no direct evidence of opioid-based treatment of hypertension is available, it is possible that both opioid agonism and antagonism could prevent the development of the disease. The inhibition of opioid systems may remove the reinforcer of blood pressure increases. However, research has also found that opioid *agonism*, such as the chronic administration of spiradoline (a nonpeptide kappa agonist) in rats, may also prevent the development of hypertension (Wright & Ingenito, 2001). In their study, hypertension-prone rats were put into a socially isolated environment, a known anxiety and stress producer. Nontreated rats developed hypertension in the stressful environment while those treated with spiradoline did not. This treatment may work either by minimizing experienced stress and thus preventing large blood pressure increases or by creating a chronically elevated opioid environment in which larger spikes of opioid

activation are impossible. In both cases, the reinforcer is removed, thus preventing learned conditions of hypertension.

The susceptible individual

A number of factors may predispose an individual to comorbid hypertension-repression conditions. Cognitive threat assessment leads to sympathetic activation which increases blood pressure, activating baroreceptors which cause the release of endogenous opioids. At any point in this process, a hypersensitive system could create the possibility of learned hypertension. An individual may be predisposed to interpret situations as threatening. Personality types that are associated with hypersensitive threat assessment may play a role here. The cardiovascular system itself may be excessively reactive. Cardiovascular reactivity is already considered to be a risk factor for hypertension (Gerin et al., 2000). A meta-analytic review of hypertension and cardiovascular reactivity to acute stressors concluded that hypertensives usually exhibit larger blood pressure responses to laboratory stressors and take longer to recover from those stressors (Fredrikson & Matthews, 1990). Certainly, if hypertension is a learned response to stressful situations, those individuals exhibiting the most variability in blood pressure and the greatest reactivity to stressors would be the ones most likely to experience hypertension as a conditioned response.

Likewise, baroreceptor sensitivity may vary in individuals. Similar to a labile cardiovascular system, a sensitive baroreceptor system would likely produce more pronounced endogenous opioid effects and thus increase the chances of a conditioned cardiovascular response. Baroreceptor reactivity has already been implicated as a possible risk factor for hypertension (Ditto & Blaine, 1990).

Environmental factors and experience with repeated stressors is another consideration. While reactivity may be an individual trait contributing to hypertension, it would also be important for an environment to provide sufficient stressors for such learning to occur, indicating that environmental factors would play an important role in the development of hypertension (Manuck et al., 1993). Development of hypertension and the repressive coping style may result from interactions between susceptible individuals and aggravating environments. Indeed, research has suggested that some importance differences are found only when critical person/situation interactions are introduced, such as the presence of a harassing confederate during an anagram task performed by individuals scoring high in hostility (Saurez & William, 1989) or high versus low control conditions presented to borderline hypertensives (Bohlin et al., 1986). Therefore, hypertension as a conditioned stress response may only occur when a vulnerable individual is placed in a stressful environment.

Limitations

Although the present study employed a strong experimental design for detecting effects of naltrexone administration, several factors potentially limited effect sizes observed in the present study. These factors will now be discussed.

Subclinical hypertension levels

The strength of between-group main effects and interaction effects may have been reduced by the use of relatively healthy college students for the study sample. Instead of using truly hypertensive sample, the study used high and low levels of a normotensive range. While the two groups were truly distinct in their blood pressure levels, differences may have been even greater if a clinically hypertensive sample was used. Previous

studies observing a comorbid hypertension/repression phenomenon have consisted of individuals with greater elevations of blood pressure than those used in the present study (e.g., Winkleby, Ragland, & Syme, 1988; Sapira, Scheib, Moriarty, & Shapiro, 1971). Future studies may make use of individuals with clinically elevated blood pressure, rather than just high normotensives. Despite the problem of using a range of normotensives, previous studies employing this design with experience of pain have produced significant results, suggesting that the effect is measurable, but perhaps limited, in a normotensive sample (Breuhl, Carlson & McCubbin, 1992).

Subclinical repression levels

Likewise, the strength of the present study may have been reduced by the use of subclinical repressive individuals. Just as antidepressants are unlikely to produce large changes in nondepressed individuals, we are unlikely to see the "treatment" naltrexone to have any significant effect on normal levels of repression. If the drug were administered to those presenting with pathologically high levels of repression, a stronger effect might be observed. Future studies investigating the role of endogenous opioids in repressive coping might make use of clinically repressed individuals.

Small sample size

While the sample size used in the present study was adequate for within-subjects tests, there was little power for testing between-groups effects. Although hypotheses involving within-subjects variables were supported, group differences were not found in most cases. These differences may reach significance with a larger sample size.

Effect of repeated measures

The present study employed four identical sessions to measure within-subjects effects. While this provides maximum control over potentially confounding factors, it produces another problem: autocorrelation. Autocorrelation has the potential to inflate and deflate statistical differences; however, in the present design, the effect was more likely to reduce variability between sessions. As subjects completed the exact same set of questionnaires in each session, there is the likelihood that responses in later sessions may be more influenced by responses in previous sessions than in present mood. While the scales in the packets were in different orders in each session, the "momentum" produced by previous completions of the scales may carry through future sessions, and produce similar responses. At least one participant claimed that he had "memorized" the responses by the fourth session. The temporal proximity of the sessions may have exaggerated this effect. Forty-eight hours was the minimum time allowed between sessions. This time was chosen to allow for adequate elimination of the active drug (complete elimination is usually achieved by 24 hours); however, responses to a scale might be remembered over a two-day period. Although participants were encouraged to "answer each item as honestly as possible," and to "answer the items as they feel now," there is no guarantee that individuals did not use schemata set by the first session to answer scales in subsequent sessions. Alternatives, such as using different measures for each session, or less objective measures, would present other flaws in the design. Future studies may minimize the effect of repeated measures by increasing the time between sessions and using only two sessions (rather than the four in the present design).

Acute administration of the active pharmaceutical

While the mechanisms that might lead endogenous opioid antagonism to affect cognitive events such as repression are unknown, it is possible that these mechanisms are not easily engaged in an acute fashion. Where complex cognitive styles, defenses, and coping mechanisms are involved, some time may be required before these systems "reorganize" themselves around the new physiological "environment." To return to the anti-depressive metaphor, it is unlikely that an anti-depressive medication would have an effect with one administration. Usually, sustained administration over a long period of time is required before changes in mood and cognitive styles can be observed. A coping strategy that has been developed over years may be initially resistant to any physiological change; therefore, results achieved with an acute administration of a drug may be minimized. In regard to endogenous opioid activity and repression, large changes in coping style might be observed only with daily administration of the naltrexone. Several factors make this design difficult however, such as low adherence (especially considering the potential negative side-effects) and the low but possible chance of liver damage (in susceptible individuals). While long-term administration may produce theoretically interesting changes, there is little feasibility in using naltrexone as a sustained treatment for repression. The practical usefulness of naltrexone lies in its ability to produce acute changes in repressive symptoms. However, if hypertension precedes repression, direct treatment of the hypertension may be a more promising route of treatment. As evidenced by the systolic increases in the present study, treating repression without treating hypertension may lead to even higher increases in blood pressure.

Short administration-to-measurement interval

In a similar way, the short amount of time between administration of naltrexone and measurement of dependent variables may have contributed to the small observed effects. While peak plasma absorption is usually achieved by one hour (Meyer, Straughn, Lo, Schary, & Whitney, 1984), it is unknown whether this is adequate time for cognitive effects to occur. Absorption time may vary by individual and is likely impacted by factors such as prior food consumption. Since naltrexone's potential impact on cognitive functions have never been assessed, it in unknown by what exact mechanisms the change occurs and how long it takes for this change. While the present study certainly shows significant effects after one hour, it is possible that a longer administration-measurement interval would yield larger results. As evidence for this possibility, participants were asked to guess whether they have received the active drug or the placebo. Several participants who responded "placebo" later returned and said they wanted to change their original guess to "drug," as effects surfaced later in the day after the session has ended. Of the four times this occurred, the newer guess of "drug" was always correct. This suggests that significant experienced effects of the drug may not manifest until some time has passed. While this possibility was noted prior to the running of the study, alternatives were determined to be too problematic to be helpful. Scheduling problems and adherence problems would have been greatly increased by requiring participants to stay, for example, two hours while the drug is absorbed. Furthermore, due to possible serious side effects (such as anaphylactic shock) which usually manifest in the first hour, it was considered unsafe to allow participants to consume the drug and then return later for measurement. In cases where human subjects are used, participant safety is paramount to

design concerns and proper safety would require monitoring during the administration period.

Incomplete antagonism of opioid and non-opioid systems

While naltrexone is an extremely effective opioid antagonist, its affinity is roughly 10 times greater for *mu* receptors than *kappa* receptors (Preston & Bigelow, 1992; Martin, 1984). Therefore, *kappa*-mediated effects may still be expressed despite complete blockage of *mu* effects. Some parts of the opioid-analgesic system may still be operating in the presence of naltrexone, which would further serve to diminish observed differences between the drug and placebo. While the *mu* receptors seem important in the role of analgesia of physical pain, it is not known how the receptors differentially mediate the cognitive effects of opioids in regard to repression. There is evidence, however, that *kappa* antagonism actually increases *dysphoric* effects (see Schlaepfer, Stain, Greenberg, Preston, & Lancaster, 1998), suggesting *kappa* antagonism may not further diminish repressive phenomena. Study of *kappa* antagonism and its effect on pain and repression is greatly hindered by the lack of *kappa* antagonists although it is generally thought that *kappa* activity is unrelated to euphoria (Julien, 1992).

Furthermore, research in hypertension/repression mechanisms is complicated by the existence of *non-opioid* intrinsic analgesic systems (Terman, Shavit, & Lewis, 1984). The existence of pain systems other than opioid-mediated ones is supported by research showing that some forms of analgesia can be reliably blocked with naloxone while others cannot (Cannon et al., 1982) and that the relationship between blood pressure and pain perception is not always moderated by the introduction of naloxone (McCubbin & Bruehl, 1994). Both systems of analgesia (opioid and non-opioid) have been selectively

activated by varying, with rats, shock temporal patterns (Lewis, Cannon, and Liebeskind, 1980), body region of the shock (Watkins & Mayer, 1982) and escapability of the shock (Moye et al., 1983). While the mechanisms involved in the non-opioid system(s) are not clear, there are suggestions that histamine (Terman, Shavit, & Lewis, 1984; Terman, Lewis, & Liebeskind, 1982; Lewis, Terman, Nelson, & Liebeskind, 1984), serotonin, or vasopressin (Randich and Maixner, 1984), may play a role.

If the inhibition of pain is a complicated collection of multiple opioid and non-opioid systems, it is most likely that mechanisms for cognitively-experienced stress is just as complicated. Therefore, the selective opioid antagonist naltrexone may only hinder part of this system. Larger effects on repressive phenomena may be observed when multiple routes of antinociception/analgesia are manipulated.

Conclusion

Taken together, the results of the present study make a convincing argument for the role of endogenous opioids in hypertension and repression. While certainly exploratory in nature, it provides initial evidence that endogenous opioids may have much more important implications to humans than just analgesia. Endogenous opioids may indeed play a critical role in both the development of repression and hypertension.

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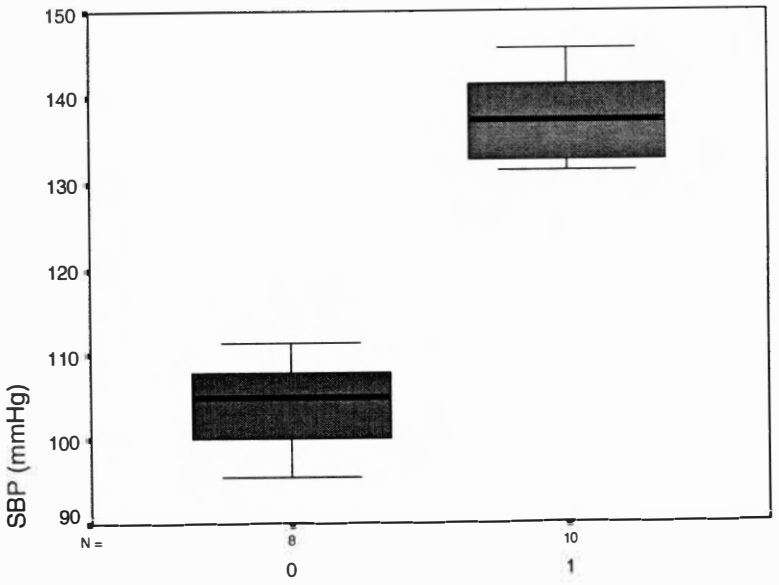
APPENDIX

a)



b)

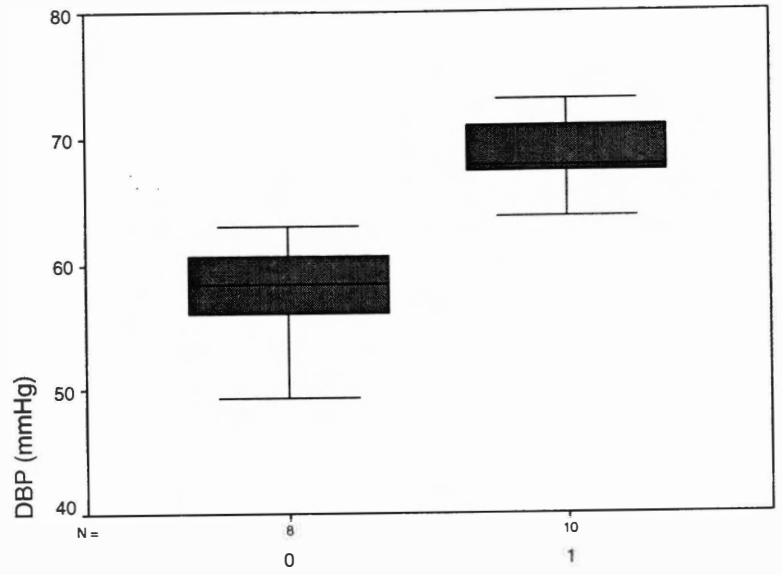


FIGURE A-1: Group differences in a) SBP and b) DBP in the cardiovascular status confirmation (validation)

TABLE A-1: SBP and DBP for participants in the initial screening and confirmation

	a) Initial Screening		b) Confirmation	
Subject	SBP	DBP	SBP	DBP
Low pressure group				
2	108	52	107	63
3	102	55	104	57
6	95	58	102	55
8	98	56	106	58
13	103	59	98	62
14	98	53	111	58
16	99	49	95	60
17	107	57	109	49
High pressure group				
1	135	67	135	65
4	135	65	131	67
5	142	74	141	73
7	146	60	139	67
9	139	63	141	72
10	140	63	131	68
11	141	60	133	64
12	135	53	146	68
15	140	74	139	71
18	132	69	135	68

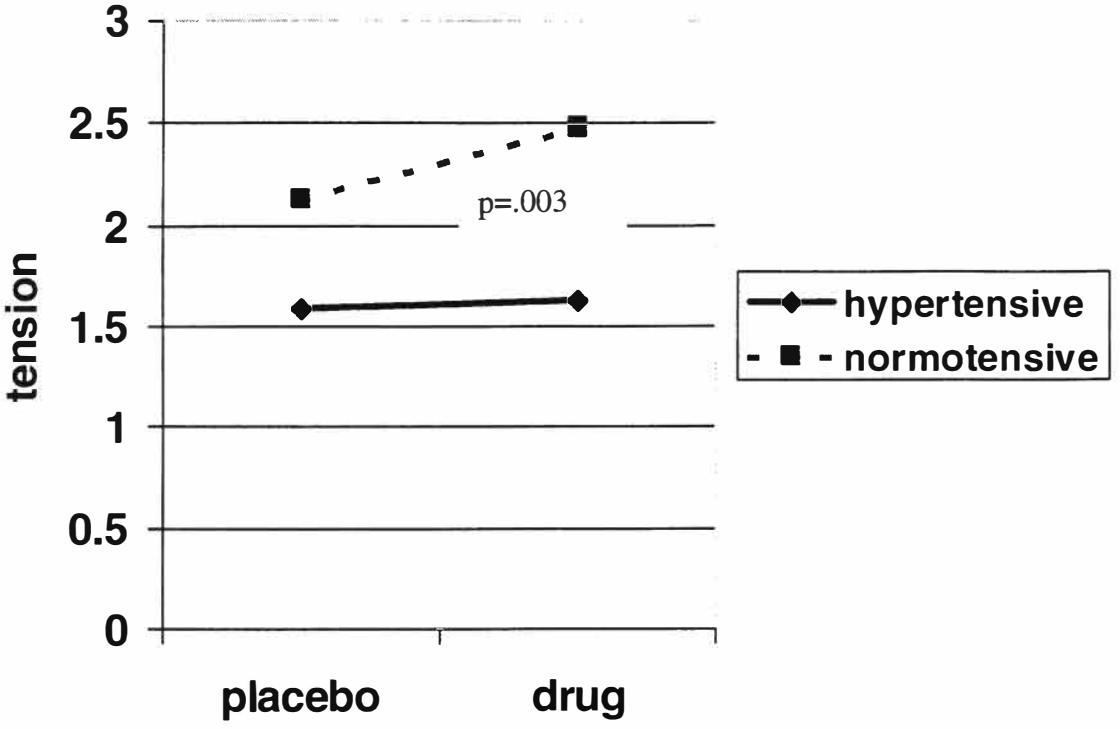


FIGURE A-2: Effect of drug on POMS tension responding for both hypertensives and normotensives

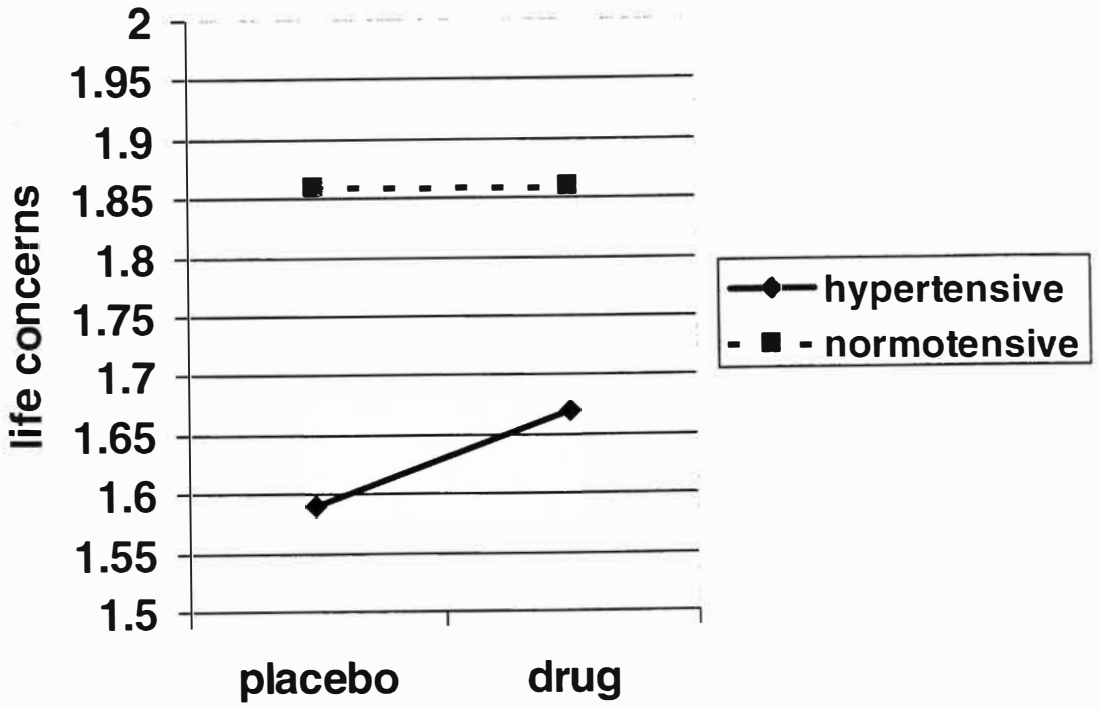


FIGURE A-3: Effect of drug on life concerns for hypertensives and normotensives

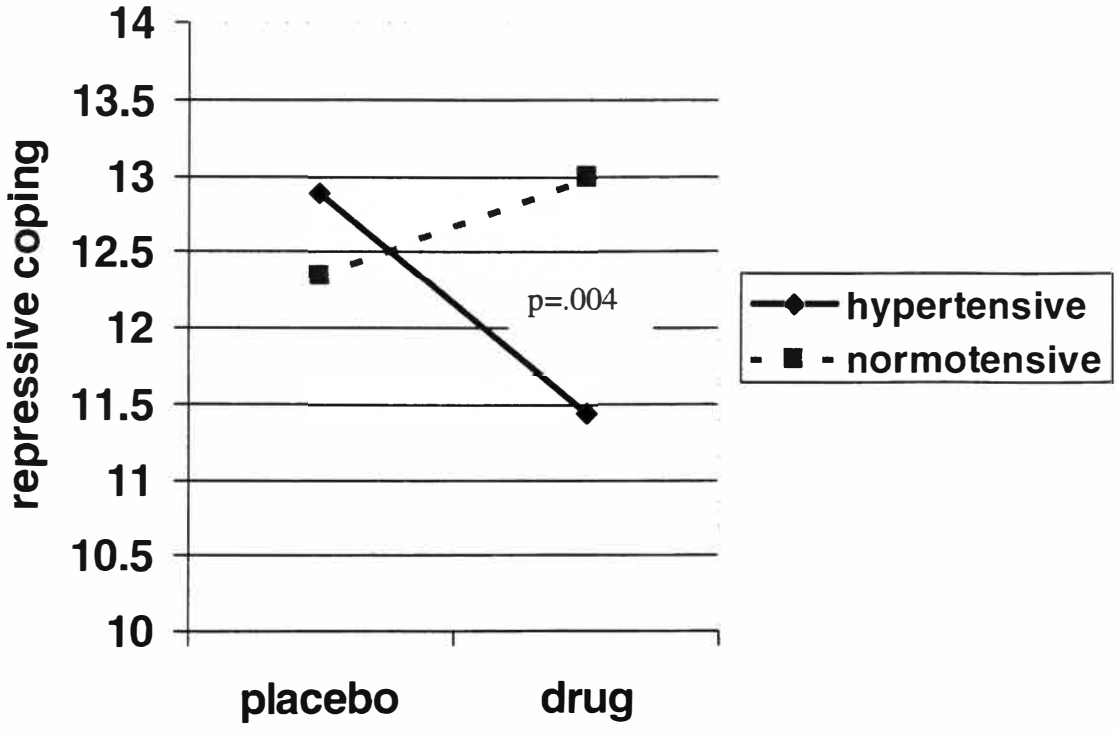


FIGURE A-4: Repressive coping as measured by the ISE for hypertensives and normotensives in the drug versus placebo conditions

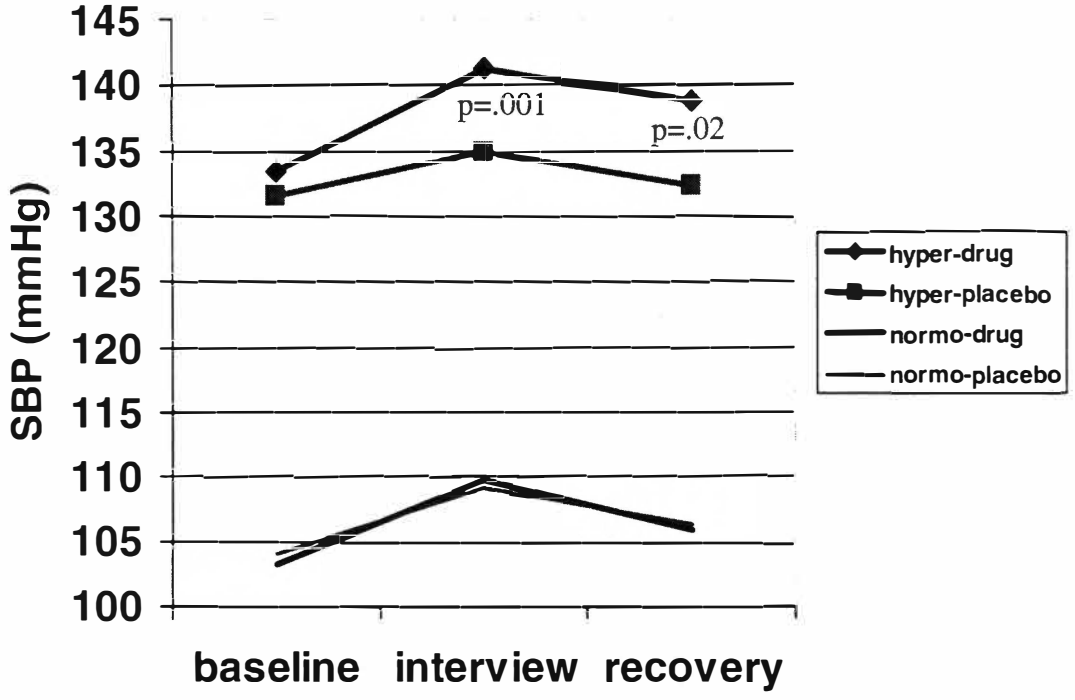


FIGURE A-5: SBP over baseline, interview, and recovery for hypertensives and normotensives in drug and placebo conditions

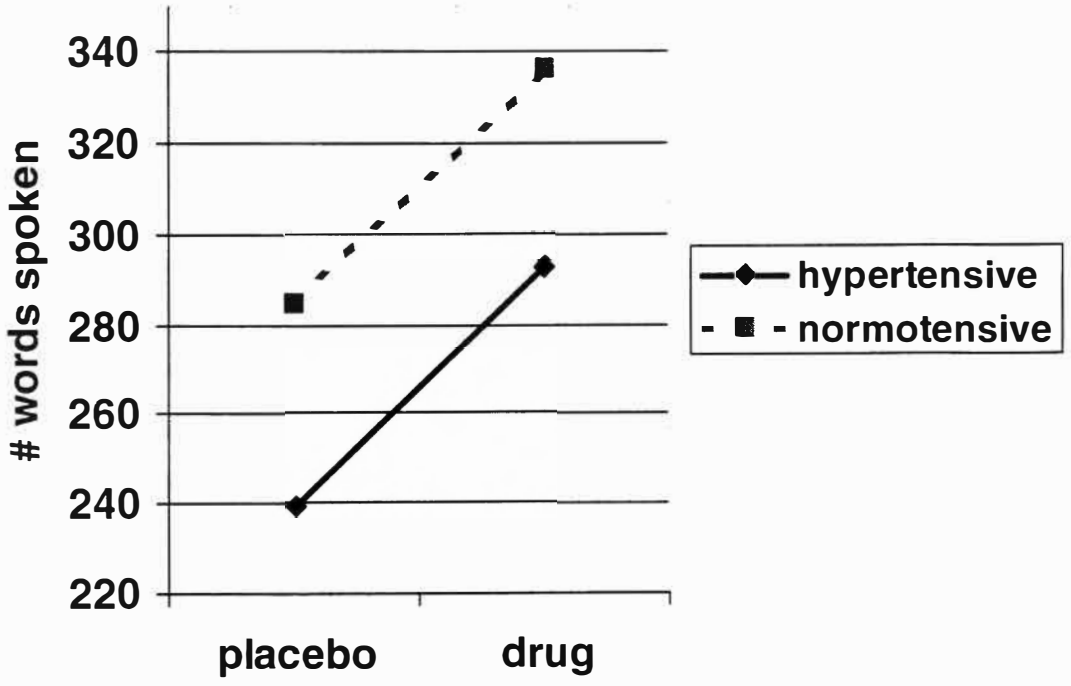
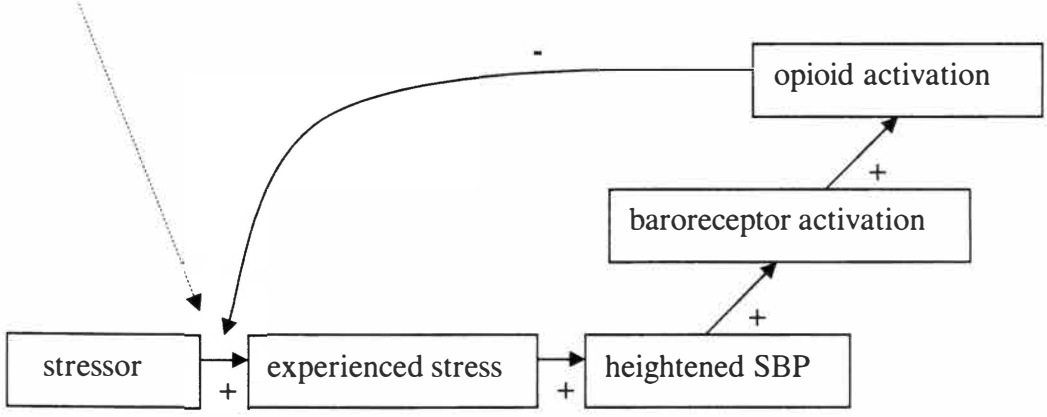


FIGURE A-6: Number of words spoken as a function of drug condition

a) without inhibition of opioid systems:

opioid activity moderates the relationship between objective stressor and subjective stress by attenuating cognitive appraisal of threat



b) with inhibition of opioid systems:

naltrexone blocks the inhibitory effects of opioids; therefore, blood pressure levels rise uninhibited

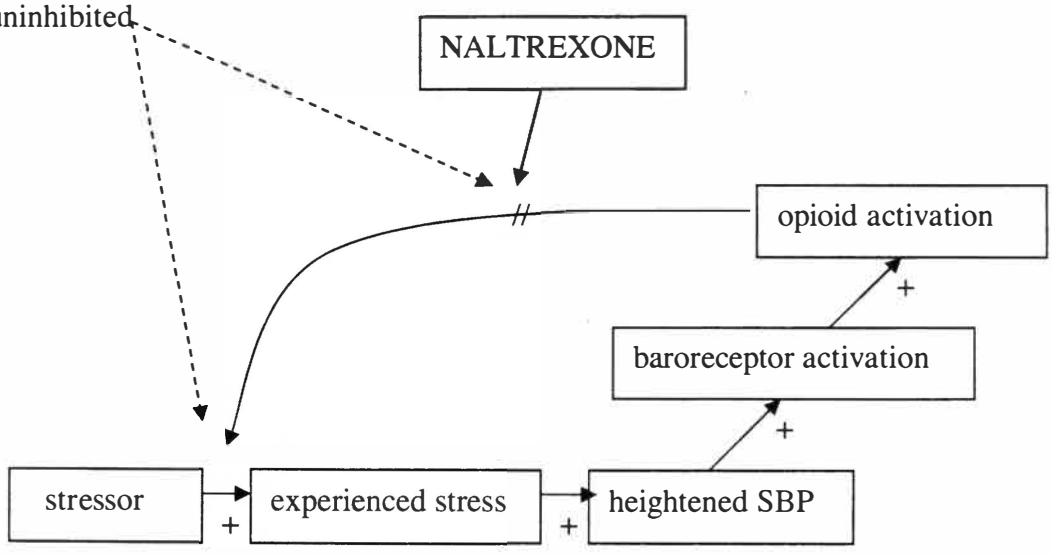


FIGURE A-7: Endogenous opioid feedback loop (a) normal and (b) disrupted with naltrexone

Vita

Jarred Wayne Younger was born in Dyersburg, TN on February 7, 1976. He was raised in Townsend, TN and went to school at Townsend Elementary, Walland Middle, and Heritage High schools. He graduated from Maryville College in 1998 with a major in Psychology. From there, he went to the University of Tennessee, Knoxville and is currently pursuing his doctorate in Experimental Health Psychology.